point indicating polyps is achlorhydria, whether in the absence or in the presence of the foregoing distinguishing features. Duodenitis would appear to be the only inflammatory condition capable of simulating hyperplasia of Brunner's glands. Duodenal inflammatory changes *per se* rarely occur. It might be more difficult to differentiate duodenitis from hyperplasia if changes in the pattern of the gastric mucosa suggest the probability of a similar pattern in the duodenum. The following signs may be helpful in differentiating duodenitis from hypertrophy of Brunner's glands: (a) bulbar irritability, with or without spastic deformity, (b) inconstancy in size and shape as well as position of the radiologic defects, (c) obliteration of the radiolucencies on compression and (d) regression of the x-ray manifestations on therapy. It is well to remember that unless hyperacidity is present, hyperplasia of Brunner's glands is unlikely.

SUMMARY

A case is reported of radiologically proved Brunner's glands hyperplasia. This diagnosis can be made in a patient with a history of dyspepsia, with gastric hyperacidity and in whom multiple radiologic defects with a nodular pattern can be observed.

Conservative treatment with diet, antacids and anticholinergic drugs appears to be quite satisfactory. Surgical intervention does not seem to be indicated, since the hypertrophic glands appear to afford a protective mechanism against the action of hydrochloric acid, and perhaps more important, since the duodenal changes in this entity may be regarded as a benign process.

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High Pressure Patent Ductus Arteriosus

A Report of Three Cases

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MOST CASES of patent ductus arteriosus are characterized by a machinery-like murmur heard during systole and diastole at the left second intercostal space. An x-ray film of the chest shows engorged lung fields, a prominent pulmonary artery segment and an enlarged ascending aorta. Electrocardiographic tracings give evidence of left ventricular strain and hypertrophy, followed, in the more pronounced cases, by right ventricular hypertrophy. A definitive diagnosis can usually be made by cardiac catheterization showing an increase in the oxygen content of the pulmonary artery. The increase is usually most pronounced in the left pulmonary artery.

The following report is based on the diagnosis and surgical treatment of three patients with pronounced pulmonary hypertension due to a patent ductus arteriosus. The pressure in the pulmonary circulation in two of these patients was as high as it was in the systemic circulation, and in one it was two-thirds as high as the systemic pressure.

REPORTS OF CASES

CASE 1. The patient was a girl 6 years of age. At the age of 18 months the mother noted that the child became very tired, especially at the end of the day. She was short of breath. At the age of two years, the patient complained of substernal pain which was intermittent and unrelated to exercise. The child had turned blue on occasion. She had frequent respiratory tract infections and was undernourished and ate very poorly. The patient had been receiving digitalis for heart failure for several years.

On physical examination the heart was observed to be very much enlarged, the left border being at the anterior axillary line in the sixth intercostal space. There was usually only a grade IV systolic murmur but at times a grade II diastolic murmur also could be heard at the left second and third intercostal spaces. P_2 was three times as loud as A_2. The blood pressure was 130/0 of mercury. The hemoglobin content was 11.8 gm. per 100 cc. of blood. Erythrocytes numbered 4.2 million and leukocytes 10,850 per cu. mm. No abnormality was noted in the urine. In an x-ray film of the chest, pronounced right and left ventricular enlargement were observed. The pulmonary vessels were engorged and pulsated actively. The pulmonary outflow tract was prominent. An electrocardiogram was interpreted as showing left atrial and left ventricular enlargement.

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Cardiac catheterization was performed and the femoral artery pressure was 135/55 mm. of mercury with a pulmonary artery pressure of 80/50 mm. The right ventricular pressure was 80/4 mm. There was a 4 volumes per cent increase in oxygen saturation of the blood in the pulmonary artery compared with the atrial specimens. Just beneath the valve in the right ventricle there was a 2 volumes per cent increase in oxygen saturation of the blood as compared with the rest of the right ventricular specimens. A patent ductus arteriosus was visualized in a retrograde aortogram.

On October 24, 1957, the patent ductus arteriosus was divided. It was 1.5 cm. in diameter and 0.75 cm. in length. The patient gained weight after the operation and, in contrast to her preoperative status, was able to play with friends without tiring. X-ray films showed a decrease in the cardiac silhouette and in the pulmonary vascular markings. The edge of the liver, palpated five fingerbreadths below the costal margin before operation, was felt at the costal margin after the patient recovered.

CASE 2. The patient, a girl 4 years of age, had had pneumonia when 18 months old, and the mother was informed at that time that the patient had "a heart murmur." The patient frequently had respiratory tract infections and exertional dyspnea. She had never been digitalized.

On physical examination it was noted that the heart was enlarged to the anterior axillary line at the sixth intercostal space. There was a grade VI systolic thrill and a typical machinery-like murmur heard at the left second and third intercostal spaces. At times, however, only a systolic murmur could be heard. The edge of the liver was felt four fingerbreadths below the right costal margin.

The hemoglobin content was 13.8 gm. per 100 cc. of blood. Erythrocytes numbered 4.7 million and leukocytes 10,700 per cu. mm. Results of urinalysis were within normal limits. Fluoroscopic examination showed the heart to be enlarged anteriorly and posteriorly to the right, and also to the left. The pulmonary arteries and peripheral pulmonary vessels pulsated vigorously and appeared to be more prominent on the right than on the left. An electrocardiogram was interpreted as showing left atrial enlargement and a suggestion of right ventricular hypertrophy.

Right heart catheterization was done on January 29, 1958. The right ventricular pressure was 145/4 mm. of mercury. The catheterization did not reveal any evidence of a ventricular septal defect. As the catheter could not be passed into it, pulmonary artery specimens were not obtained.

The ductus was divided on February 25, 1958. It was 12 mm. in diameter and in length. The edge of the liver, which was four fingerbreadths below the costal margin before operation, was three fingerbreadths below immediately afterward, and six hours later it could not be palpated. The patient did well after recovery. When last observed she was playing normally and was gaining weight.

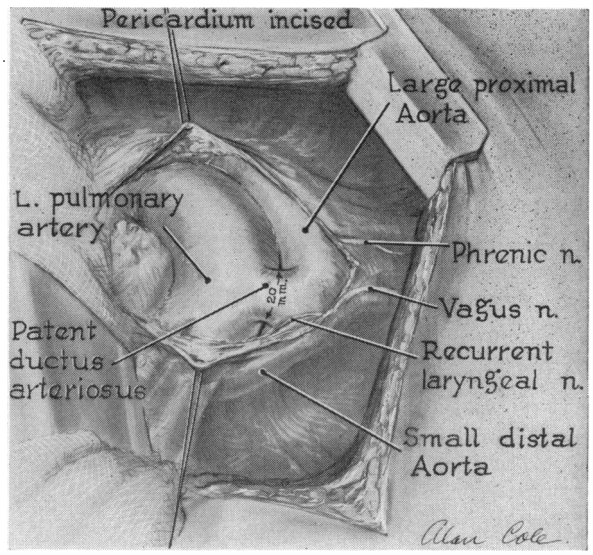

Figure 1.—Artist's drawing of large patent ductus arteriosus in Case 3. The pulmonary artery pressure was equal to systemic artery pressure.

CASE 3. The patient was a 12-year-old girl who was reported to have had a "heart murmur" first noted at the age of six weeks. Cyanosis was noted at the age of six years on a hot day after the patient had been playing very strenuously, and again at the age of eight years. She usually rested in a squatting position up to the age of eight years but not after that. Shortness of breath on exertion was frequently noted. The patient had been receiving digitalis for four years.

The heart was enlarged to the anterior axillary line in the sixth intercostal space. P_2 was four times as loud as A_2. There was no diastolic murmur. The liver was palpated two fingerbreaths below the costal margin.

Cardiac catheterization was carried out. The pressure was 130/0 mm. of mercury in the right ventricle and 130/80 mm. in the pulmonary artery. The oxygen saturation of the blood in the pulmonary artery was four volumes per cent higher than in the blood in the right ventricle.

On March 5, 1958, the ductus was divided (Figure 1). It was 20 mm. in diameter and 5 mm. long. The patient did very well and returned to full activity.

OPERATIVE TECHNIQUE

The technique used in these cases was as follows:

The patient was placed in the posterior lateral position and entrance into the chest was made through the fifth intercostal space on the left side. The ductus arteriosus was isolated. Care was taken not to injure the vagus nerve, the phrenic nerve or the recurrent laryngeal nerve. Potts clamps were placed on the aortic side of the ductus and on the pulmonary artery side of the ductus. The ductus was partially divided and a 4-0 arterial silk mattress

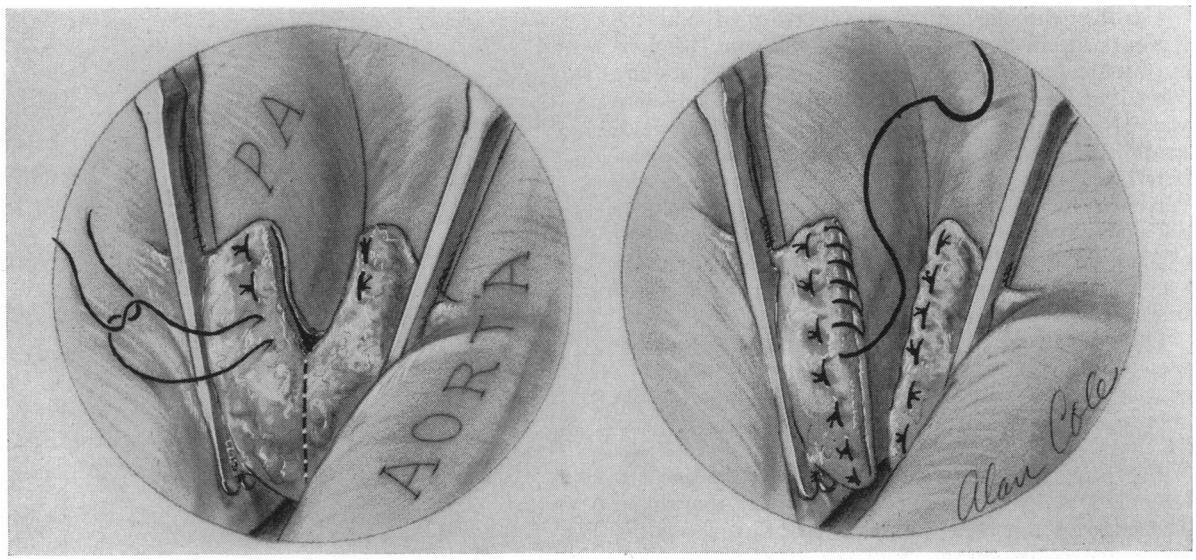

Figure 2.—Safe method of division to patent ductus arteriosus.

suture was placed on the severed end of the ductus near the aortic side and another similar suture was placed on the pulmonic side. Further division of the ductus was then carried out and more mattress sutures were placed on each side. The procedure was continued until the ductus was completely divided. Then each end of the ductus arteriosus was further secured by two rows of continuous 5-0 arterial silk (see Figure 2). This technique is essentially that described by Bosher[1] for the safe division of a patent ductus arteriosus.

DISCUSSION

The first successful operative procedure for patent ductus arteriosus was performed in 1939 by Gross.[3] Since then, the technique has been improved by various surgeons. At present, most investigators agree that all patients with patent ductus arteriosus and a left to right shunt should have surgical correction of this lesion. The operative mortality is lower than the risk of subacute bacterial endocarditis and heart failure. The operative mortality is less than one-third of 1 per cent.

The diagnosis of patent ductus arteriosus is not difficult if the typical murmur is present. However, Damman and Sell[2] in 1952 reported that patients with high pressures in the pulmonary artery caused by a patent ductus arteriosus may have only a systolic murmur.

Cardiac catheterization should be done in all cases in which the diagnosis is not definite. However, at times it may be impossible even with catheterization to distinguish between a ventricular septal defect, an aortic septal defect and patent ductus arteriosus. Because of this, in the first of the three cases herein reported it was necessary to carry out retrograde aortography by inserting a needle into the femoral artery, passing a catheter through the needle into the ascending aorta and injecting radiopaque dye while taking multiple films rapidly. This technique outlined the patent ductus arteriosus very well and differentiated this lesion from a ventricular septal defect and an aortic septal defect.

SUMMARY

Three patients with patent ductus arteriosus in whom, at cardiac catheterization, the pressures in the pulmonary artery were extremely high, are presented. In two patients the pressure was as high in the pulmonary artery (130 mm. of mercury) as in the systemic circulation, and in the third patient the pressure was two-thirds as high. The patients all were greatly improved after the ductus was divided.

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